Reference to this paper should be made as follows:


**Abstract**

W.L.H. chicks were infected with A. galli eggs low dose (500 eggs) and cadmium acetate treatment (5 mg. / 100 ml.) was given subsequently. Bursa showed Various pathological changes, after 30 days of Post infection and treatment. Majority of lymphocytes in bursal follicles showed retrogressive changes and size of follicles found to be much atrophied. The irregular inflammatory and non-inflammatory edema and depletion of lymphocytes were observed in present study.

**Key words** - W.L.H. Chicks, Bursa of Fabricius, Ascaridia galli, Cadmium acetate.
Introduction

The poultry sector in India has undergone a paradigm shift in structure and operation. A significant feature of India’s poultry industry has been its transformation from a way backyard activity into a major commercial activity in just about four decades. Nematode infections in man and domestic animals have been reported to be hyperendemic, sometimes to the extent of 60-80 percent (Chowdhry, 1978). *A. galli* also leads to malnutrition in chicks which results in the decreased return of products derived from poultry (WHO, 1967). Immunopathological lesions in infected and treated chickens are formed resulting into hypoplasia and atrophied bursa of fabricius (Bagust *et al.* 1979, Taniguchi *et al.* 1977, Alzeter *et al.* 2006). Larval stages of *A. galli* of chicks causes anaemia, hyperproteinemia and other hyperplastic pathological disorders including haemaorhagic inflammation associated with edema. According to Lloyd (1978) the complete eradication of these parasitic disease would be of major health importance. Cadmium also produces immunotoxicity (Descotes 1992) Pollutants or any environmental stress affect haematological, biochemical and immunopathological parameters.

Materials and Methods

Just hatched, healthy white leg horn (WLH) chicks were obtained and kept in spacious wooden cages under room temperature, sufficient aeration and suitable light. They were fed on formulated chick feed of Hindustan Poultry Feed Ltd., India. The inocula with desired numbers of embryonated eggs. (500 embryonated eggs) were administered orally to chicks. Chicks were maintained for 30 days of exposure and bursa tissues were obtained for immunopathological studies.

The dose with desired amount of cadmium acetate (5ml/100ml) was prepared and administered orally to chicks for immunopathological studies chicks were divided into following groups -

- **Group I** - Healthy chicks (control) - 12 chicks.
- **Group II** - Chicks infected with 500 embryonated eggs of *A galli* - 12 chicks.
- **Group III** - Chicks infected with 500 embryonated eggs of *A galli* and cadmium acetate treatment - 12 chicks.

Chicks were anesthetized, decapitated and autopsy was performed. Bursa was removed and placed in 10% neutral buffer formalin. All organ sample were processed by standard histopathological techniques. Sections were stained with hematoxylin and eosin Microphotography of sections were done.
Pathological study of Primary Lymphoid Organ (Bursa of Fabricius) in W.L.H. chicks along with Experimental Ascaridiasis

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Results

**Group 1** Structure of bursa of healthy chicks (control) Fig. 1, 2

The transverse section of bursa from control chicks showed the following structure. The bursa was broadly divided into...
regions (1) capsule  (2) Sub capsule.

**Capsule** - It was made up of outer thin serosa layer and inner muscularis. The outer most layer of bursal capsule constituted serosa which surrounded the entire capsule. The inner layer, muscularis constituted major portion of capsular wall.

**Sub capsule** - It consisted of mucosa and surface epithelium. The mucosa developed into a number of villus like projections invading the central lumen, called plicae. Each plicae consisted of a number of polyhedral follicles, closely packed together with small amount of connective tissue separating them and blood vessels lying between them. Each follicle was divided into two regions (a) outer cortex (b) inner medulla.

Both cortex and medulla possessed a supporting network of stellate reticulo-endothelial cells whose meshes were filled with lymphoid cells.

**Group II** Structure of bursa of chicks infected with 500 embryonated eggs. (Fig. 3,4) Histologically, the capsular wall revealed slight thickening. Hypertrophy of plicae was observed leading to decrease in bursal lumen. The follicles were decreased and widely separated by oedematous fluid and also revealed in varying degree distinct inflammatory edema while medulla contained less number of small lymphocytes and large number of medium lymphocytes. The plicae wall was observed to be ruptured and interfollicular space of plicae was increased. A considerable parallel type non inflammatory edema was also observed in both cortex and medulla.

**Group III** structure of bursa of chicks infected with 500 embryonated eggs and treated with Cadmium acetate (Fig. 5, 6) Hypertrophy of plicae and follicles was seen due to increase in cell population while inter follicular space of plicae has disappeared in follicles. Some follicles become separated from epithelial layer. Vacuolization was observed, with a single vacuole in each follicle due to devoid of cytoplasm. No significant changes were observed in bursal capsule. Cortex and medulla were also observed to be normal but non-inflammatory edema inside some follicles could be observed. There was a clear evidence of rupture of follicles while follicles showed presence of numerous lymphocyte cells.

**Discussion**

In the present investigation, it has been revealed that experimental *A. galli* infection and cadmium intoxication induced marked array of pathology of bursa of fabricius in W.L.H. chicks. Histologically, capsular wall was found to be more or less thickened. The follicles were found to be atrophied. The irregular inflammatory and non-inflammatory edema and depletion of lymphocytes were observed in present study. Lymphoid follicle atrophy was probably the result of migration of lymphocytes to the site of infection. It may be attributed to the leakage of endotoxins into the bursa, released during antigen-antibody interaction. It may be due to severity of infection and intoxication.
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Bursal lesions have been observed by Faragher (1972), Okoyo (1984), Mohanty and Rao (1984) and Mishra (1984). Akter et al. (2006) observed histopathological changes in lymphoid tissue analyzed in broiler chickens. Oznurlu et al. (2010) studied about histochemical and histological evaluation of effects of high incubation temperature on embryonic development of thymus and bursa of fabricius in broiler chickens. Immunopathologically, the bursa of fabricius revealed severe reterogressive changes of lymphocytes in most of lymphoid follicles and many of lymphocytes were replaced by reticuloendothelial cells. Interfollicular edema, fibroplasias associated with infiltration of lymphocytes, monocytes and plasma cells (Singh and Rao, 1987).

The follicular hyperplasia was due to increase in number and size of secondary follicles, but there was variability in degree of hyperplasia between the animals (Carpenter et al. 2007). In other circumstances the virus propagated in Vitro did not cause lymphocytosis or lymphoadenopathy (Suarez et al. 2005). Diffused lymphoid tissue was noted in the bursal canal and follicles became seperated from epithelial layer with the medulla. The epithelial layer and lumen also disappeared leaving muscle, connective tissue, blood vessel and follicles with and without medulla (Word and Midelletion, 1993). Degenerative changes and depletion of lymphoid cells were observed in bursa of fabricius of vaccinated broiler chicks (Jeurissen et al. 1998, Stoey et al. 2000).

On the basis of various experimental evidences, the above studies provided that A galli infection and toxicant induce immunopathological alteration occurred in lymphoid organs. Studies regarding pathological modulations would be helpful in understanding immune system of the host.

References:


118